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MORPHOLOGIC STUDIES

Morphologic Comparison of Frequency and Types of Acute Lesions in The Major Epicardial Coronary Arteries in Unstable Angina Pectoris, Sudden Coronary Death and Acute Myocardial Infarction

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The frequency and type of acute lesions in the four major (right, left main, left anterior descending, left circumflex) epicardial coronary arteries were examined at necropsy in 14 patients with unstable angina pectoris, 21 patients with sudden coronary death and 32 patients with a fatal first acute myocardial infarction. None of the 67 patients had a grossly visible left ventricular scar (healed myocardial infarct) and only the group with acute myocardial infarction had left ventricular myocardial necrosis.

Although the frequency of *intraluminal thrombus* was similar in patients with unstable angina (29%) and sudden death (29%) and significantly lower than in those with acute infarction (69%) ($p = 0.02$), the thrombus in the patients with unstable angina and sudden death consisted almost entirely of platelets and was nonocclusive, whereas the thrombus in the group with acute infarction consisted almost entirely of fibrin and was occlusive. The frequency of *plaque rupture* was insignificantly different in the groups with unstable angina (36%) and sudden death (19%), and was significantly lower than in the group with acute infarction (75%) ($p = 0.02$).

The frequency of *plaque hemorrhage* was insignificantly different in the groups with unstable angina (64%) and sudden death (38%) and was significantly lower than in the group with acute infarction (90%) ($p = 0.04$). The frequency of atherosclerotic plaques containing *multiluminal channels* was similar in patients with unstable angina, sudden death and acute infarction (100%, 81% and 90%, respectively), but the percent of 5-mm long segments of the four major coronary arteries containing multiluminal channels (probably the result of organization of thrombus) was greatest in the unstable angina pectoris group (12% vs. 7% vs. 1%, respectively; $p = 0.04$).

Thus, the frequency of thrombus, plaque rupture and plaque hemorrhage in the coronary arteries among patients with unstable angina pectoris and sudden coronary death was similar and significantly less than in the patients with acute myocardial infarction. The type of thrombus and the amount of lumen obstructed by thrombus were similar in the groups with unstable angina pectoris and sudden coronary death and quite different from those in the group with acute myocardial infarct group.

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In recent years, considerable effort has been directed toward understanding the acute coronary events that may be responsible for the development of unstable angina pectoris, sudden coronary death and acute myocardial infarction. From angiographic, angioscopic and necropsy studies (1–14), it has been speculated that plaque rupture and hemorrhage with overlying intraluminal thrombus, which are the acute coronary lesions usually responsible for acute myocardial infarction, are also responsible for unstable angina pectoris. Although necropsy studies in patients with unstable angina have demonstrated plaque rupture or intraluminal thrombus, or both, all reported studies have included patients who also had acute myocardial infarction or sudden coronary death, or both. Thus, it is possible that patients with unstable angina complicated by acute myocardial infarction have acute coronary lesions similar to those in patients with acute infarction not preceded by unstable angina.

Because of the intermixing of unstable angina pectoris, sudden coronary death and acute myocardial infarction in previously reported studies, we examined in detail the major epicardial coronary arteries in patients whose only manifestation of myocardial ischemia was unstable angina not complicated by acute infarction and compared the frequency and type of acute coronary lesions in these patients with those in patients with a fatal first acute myocardial infarction and victims of sudden coronary death in whom myocardial necrosis was absent.

Methods

Cases studied and selection criteria. The autopsy records of the Pathology Branch, National Heart, Lung, and Blood Institute were searched and all cases coded as fatal coronary artery disease were reviewed. Patients who had one or more

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left ventricular scars (healed myocardial infarct), had undergone coronary angioplasty or coronary bypass surgery, had received thrombolytic therapy or were ≤40 years of age were excluded.

The patients with unstable angina pectoris included those who died in the hospital (13 patients) or shortly (<1 day) after hospital discharge (1 patient); none had gross or microscopic evidence of left ventricular necrosis at necropsy. In all 14 patients, there was electrocardiographic (ECG) evidence of ischemia during the final hospital admission or chest pain at rest within 24 h of death, or both. These 14 cases included all patients studied in the Pathology Branch with unstable angina pectoris in whom coronary angioplasty or bypass surgery or thrombolytic therapy had not been performed or administered, in whom no left ventricular scars were present and in whom the 5-mm coronary sections were available for examination.

All 32 patients with acute myocardial infarction were studied from 1970 to 1980 and all had transmural necrosis (involvement of all the inner half of the left ventricular wall and all or a portion of the outer half of the wall).

All 21 victims of sudden coronary death died suddenly outside the hospital usually within 15 min of onset of chest discomfort and all within 3 h of symptom onset. All 21 patients were studied at necropsy from 1978 to 1988; 10 of the 21 had been included in a previous study (15). All 21 were randomly selected from the files of the Pathology Branch. To be included, all had had histologic sections prepared from each 5-mm long segment of the four major coronary arteries. None had gross or histologic evidence of transmural left ventricular necrosis or fibrosis.

The only manifestation of myocardial ischemia in any of the 67 study patients was that which caused death. No patient with fatal acute myocardial infarction, for example, had angina pectoris; no patient with unstable angina had acute infarction and no victim of sudden coronary death had unstable or stable angina or acute infarction at any time. No patient had necropsy evidence of valvular, congenital or other specific cardiac disease. All patients with unstable angina and acute infarction were diagnosed as such by the physicians providing their clinical care.

Coronary artery preparation and examination. In each case, the four major (right, left main, left anterior descending, left circumflex) epicardial coronary arteries were dissected from the surface of the heart and processed in an identical fashion. The arteries were decalcified, sectioned gently with minimal pressure transversely at 5-mm intervals with a sharp knife blade, decalcified again if necessary, dehydrated in ethanol, cleared in xylene and embedded in paraffin. Both a Movat-stained and a hematoxylin-eosin-stained section of each 5-mm segment were prepared.

All sections containing intraluminal thrombus, plaque rupture, plaque hemorrhage and multiluminal channels were examined by all three authors and agreed on by each. In every section, the degree of cross-sectional area narrowing was estimated by examination of histologic sections magnified 40 times (occasionally 20 times) and the narrowing categorized into five groups: 0% to 25%, 26% to 50%, 51% to 75%, 76% to 95% or 96% to 100%. The presence or absence of thrombus, plaque rupture, plaque hemorrhage and multiluminal channels was noted in each section and confirmed by all three authors (Fig. 1 and 2).

Definitions of acute coronary lesions. *Thrombus* was defined as an occlusive or nonocclusive intraluminal aggregate of platelets or fibrin, or both, with or without associated erythrocytes and leukocytes with definite mural attachment. *Plaque rupture* was defined as a defect observed on histologic sections of coronary artery cross sections extending from the luminal surface of the plaque or with thrombus over the defect. Minor surface erosions were not considered plaque rupture. *Plaque hemorrhage* was defined as extravasation of erythrocytes into the plaque. *Multiluminal channels* were defined as multiple vascular channels present in the inner (luminal) half of the plaque.

Statistics. Statistical analysis compared the frequency of each lesion in each of the three patient groups by using either a chi-square analysis or a two-tailed Fisher's exact test.

Results

Number of patients, coronary arteries and 5-mm coronary segments studied. A total of 67 patients, 268 major epicardial arteries and 3,101 5-mm segments of the major arteries were studied: 14 patients (56 arteries and 592 5-mm segments) with unstable angina pectoris; 32 patients (128 arteries and 1,530 5-mm segments) with first fatal acute myocardial infarction and 21 patients (84 arteries and 999 5-mm segments) with sudden coronary death. Certain clinical and morphologic features in these patients are summarized in Tables 1 to 3. The age of the 67 patients ranged from 41 to 82 years (mean 61); the mean age was 61 years (11 men) in the 14 patients with unstable angina pectoris, 55 years (all men) in the 21 patients with sudden coronary death and 66 years (22 men) in the 32 patients with acute myocardial infarction. Of the 67 patients, 54 (81%) were men.

Degrees of luminal narrowing. Of the 268 major coronary arteries examined, 192 (72%) were narrowed >75% in cross-sectional area by atherosclerotic plaque, including 37 arteries (14%) narrowed >95% in cross-sectional area. The percent of arteries narrowed >75% in the unstable angina pectoris, sudden coronary death and acute myocardial infarction groups was insignificantly different (80% [45 of 56] vs. 67% [56 of 84] [p = 0.07] vs. 71% [91 of 128] [p = 0.02]), but the percent of arteries narrowed >95% was significantly higher in the unstable angina than the sudden death group (25% [14 of 56] vs. 5% [4 of 84]; p = 0.001), but not significantly different from that in the acute infarction group (15% [19 of 128]; p = 0.1).

Intraluminal thrombus. The frequency of intraluminal thrombus was similar in the unstable angina pectoris and sudden coronary death groups (29% [4 of 14] and 29% [6 of

Figure 1. Photomicrographs of transverse sections of epicardial coronary artery in patients with fatal coronary artery disease. **a.** Section of the right coronary artery from a patient with fatal acute myocardial infarction and total occlusion of the lumen by thrombus (T). There is rupture of the underlying plaque with hemorrhage (H) into the plaque that is rich in pultaceous debris and contains numerous cholesterol clefts (Movat stain ×20, reduced by 15%). **b.** Section of the left circumflex coronary artery with total occlusion of the lumen by thrombus (T). Although hemorrhage (H) is present in the underlying plaque, no site of plaque rupture was identified (hematoxylin-eosin stain ×20, reduced by 15%). **c.** Section of the left circumflex coronary artery in a patient dying with unstable angina pectoris without left ventricular necrosis. The lumen (**arrowheads**) is compressed by plaque into which there has been extensive hemorrhage (H). A site of plaque rupture was identified in an adjacent section (Movat stain ×34, reduced by 15%). **d.** Section of the left anterior descending coronary artery in a patient with unstable angina pectoris with hemorrhage (H) into a lipid-rich plaque. Plaque rupture was not identified (Movat stain ×16, reduced by 15%).

21]) and significantly lower than that in the acute myocardial infarction group (69% [22 of 32]). The thrombus was nonocclusive in all 4 patients with unstable angina, in 5 of the 6 patients with sudden death and in only 4 of the 22 patients with acute infarction. The composition of the nonocclusive and occlusive thrombi also was different: the nonocclusive thrombus consisted mainly of platelets and the occlusive thrombus mainly of fibrin. Of the 32 patients with thrombus, plaque rupture was found in association with the thrombus in 17 patients (53%): in none of the 4 patients with unstable angina, in 2 of the 6 patients with sudden death and in 15 (83%) of the 22 patients with acute infarction. In the 15 patients with thrombus unassociated with plaque rupture, hemorrhage into the plaque at the site of thrombus was found in 7: in 3 of the 4 patients with unstable angina, in 2 of the 6 patients with sudden death and in 2 of the 32 patients with acute myocardial infarction.

Plaque rupture. Plaque rupture was found in 33 (49%) of the 67 patients. Its frequency was insignificantly different in the groups with unstable angina pectoris (36% [5 of 14]) and sudden coronary death (19% [4 of 21]); in both groups, the frequency was significantly less than in the group with acute myocardial infarction (75% [24 of 32]; $p < 0.02$).

Plaque hemorrhage. This was observed in 27 (40%) of the 67 patients and its frequency was significantly lower in the

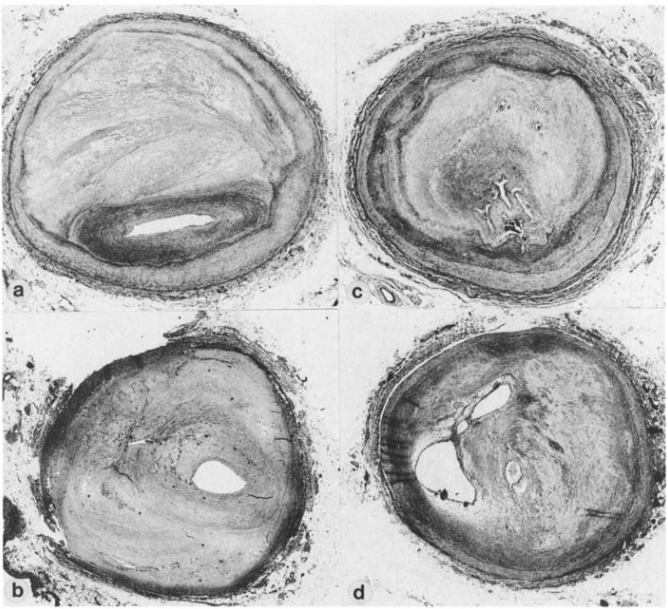

Figure 2. Photomicrographs of Movat-stained sections of an epicardial coronary artery in patients who died with unstable angina pectoris without evidence of left ventricular necrosis. **a** and **b**, Segments of a coronary artery severely narrowed by plaque composed almost entirely of fibrous tissue (Movat stain, ×27, ×26, both reduced by 15%). **c** and **d**, Segments of an artery narrowed by fibrous plaque with multiple small vascular channels (Movat stains, both ×27, both reduced by 15%).

groups with unstable angina pectoris (21% [3 of 14]) and sudden coronary death (19% [4 of 21]) compared with that in the group with acute myocardial infarction (63% [20 of 32]). Plaque hemorrhage was associated with plaque rupture or intraluminal thrombus in 20 (74%) of the 27 patients with plaque hemorrhage: in 4 of the 14 patients with unstable angina, in 4 of 21 with sudden death and in 12 of the 32 with acute infarction.

Multiluminal channels. Multiple small vascular channels were present in 60 (90%) of the 67 patients and with an insignificantly different frequency in each of the three patient groups (Table 1). The frequency of multiluminal channels in each 5-mm long segment of coronary artery was significantly higher in the group with unstable angina pectoris (12% [66 of 572]) than in either the sudden coronary death group (7% [72 of 999]) or acute myocardial infarction group (7% [107 of 1,530]) (Table 3).

Discussion

Major morphologic complications. Comparison of findings from examination of a histologic section from each of 3,101 5-mm segments of 268 major epicardial coronary arteries from 67 patients with fatal coronary artery disease disclosed that the frequency of three acute coronary lesions (namely, intraluminal thrombus, plaque rupture and plaque hemorrhage) was similar in patients with unstable angina pectoris and sudden coronary death and that the frequency of each of these acute lesions was significantly higher in patients with fatal first transmural acute myocardial infarction. Furthermore, although multiluminal channels (not acute lesions) within plaques were frequent in all three patient groups, these lesions were found significantly more often in 5-mm segments of coronary arteries in the group

Table 1. Frequency of Acute Coronary Lesions and Multiluminal Channels at Necropsy in Patients With Unstable Angina Pectoris, Sudden Coronary Death and Acute Myocardial Infarction

Coronary Subset	No. of Patients	Coronary Arteries: Thrombus	Plaque Rupture	Plaque Hemorrhage	Multiluminal Channels
Unstable angina pectoris	14	4 (29%)*	5 (36%)*	3 (21%)*	14 (100%)
Sudden coronary death	21	6 (29%)*	4 (19%)*	4 (19%)*	17 (81%)
Acute myocardial infarction	32	22 (69%)†	24 (75%)†	20 (63%)†	29 (90%)
Total	67	32 (48%)	33 (49%)	27 (40%)	60 (90%)

* versus † in same vertical column, $p < 0.02$.

with unstable angina than in the groups with sudden death and acute infarction (where their frequency was similar).

Unstable Angina Pectoris

Previous studies. Several angiographic studies (1–5) have identified either intraluminal filling defects consistent with thrombus or specific morphologic lesions (eccentric narrowings with irregular borders) in patients with unstable angina pectoris, and these defects have been used to distinguish such patients from those with stable angina (1–3). Comparison of postmortem angiographic and histologic findings (6), however, in patients with coronary artery disease (not necessarily unstable angina pectoris) has shown that these irregular eccentric lesions may represent not only sites of intraluminal thrombus, but also plaque rupture, plaque hemorrhage or organized thrombus. In addition, three angioscopic studies (7–9) have identified intraluminal thrombus and ulceration or rupture of plaque in patients with unstable angina. On the basis of these studies, it has been widely speculated that the lesion responsible for the development of unstable angina pectoris is an ulcerated plaque over which nonocclusive intraluminal thrombus develops.

Limitations of previous studies. Before accepting that this hypothesis is indeed true for all or most patients with unstable angina pectoris, the limitations of the previous studies (1–9) need to be considered. Interpretation of the significance of the eccentric irregular lesions seen angiographically in patients with unstable angina is based largely on the work of Levin and Fallon (6), who compared postmortem coronary arteriograms and histologic sections of coronary artery narrowings in 39 patients who died either after coronary artery bypass surgery or of consequences of acute myocardial infarction. (Because the trauma of bypass surgery may be associated with plaque rupture or plaque hemorrhage, or both, patients who had undergone this procedure were excluded from our study.) They identified 38 narrowings that had irregular borders or intraluminal lucencies by angiography. Of these, 8 (21%) were acute or organizing nonocclusive thrombi overlying atherosclerotic plaque, 6 (16%) were nonocclusive thrombi overlying sites of plaque rupture or hemorrhage, 10 (26%) were sites of plaque hemorrhage or rupture without thrombus, 6 (16%) contained recanalized thrombus (presumably multiluminal channels) and 2 (5%) showed narrowing of the segment by plaque without any complicating acute lesion. More than a third of the irregular eccentric lesions studied, therefore, showed no acute lesion that would account for the abrupt change in symptoms in the setting of unstable angina. In our study, plaques containing multiluminal channels, although common to all three groups of patients, were seen with greatest frequency in the group with unstable angina.

When interpreting reports of angiographic or angioscopic studies in patients with unstable angina pectoris, it is assumed that the patients did not have left ventricular necrosis (acute myocardial infarction) at the time of study, an assumption that may or may not be true. Guthrie et al. (10) studied 12 patients with unstable angina who died shortly after coronary artery bypass surgery. At autopsy, 4 of the 12 patients had acute myocardial infarction that histologically appeared to have occurred before the operation and acute myocardial infarction was not suspected clinically in any of

Table 2. Frequency of Acute Lesions and Multiluminal Channels at Necropsy in the Four Major Epicardial Coronary Arteries in Unstable Angina Pectoris, Sudden Coronary Death and Acute Myocardial Infarction

Coronary Subset	No. of Coronary Arteries	Coronary Arteries: Thrombus	Plaque Rupture	Plaque Hemorrhage	Multiluminal Channels
Unstable angina pectoris	56	4 (7%)	9 (16%)	15 (27%)	32 (57%)
Sudden coronary death	84	6 (7%)	4 (5%)	10 (12%)	32 (38%)
Acute myocardial infarction	128	22 (17%)	27 (21%)	47 (37%)	69 (54%)
Total	268	32 (12%)	40 (15%)	72 (27%)	133 (50%)

Table 3. Frequency of Acute Coronary Lesions and Multiluminal Channels at Necropsy in 5-mm Long Segments of the Four Major Epicardial Coronary Arteries in Unstable Angina Pectoris, Sudden Coronary Death and Acute Myocardial Infarction

Coronary Subset	No. of 5-mm Coronary Segments	Coronary Arteries			
		Thrombus	Plaque Rupture	Plaque Hemorrhage	Multiluminal Channels
Unstable angina pectoris	572	11 (2%)	7 (1%)	24 (4%)	66 (12%)*
Sudden coronary death	999	6 (0.6%)	5 (0.5%)	19 (2%)	72 (7%)†
Acute myocardial infarction	1,530	105 (7%)	59 (4%)	117 (8%)	107 (7%)‡
Total	3,101	122 (4%)	71 (2%)	160 (5%)	245 (8%)

* versus † in same vertical column = $p < 0.004$.

the 4 patients. Therefore, when studying living patients, it may be difficult to determine whether the patients have pure unstable angina pectoris or have combined unstable angina pectoris and acute myocardial infarction.

Information regarding coronary artery morphology in patients with unstable angina pectoris is scant, difficult to obtain and difficult to interpret for several reasons. Unstable angina pectoris is rarely fatal and those patients who do die during the period of unstable angina usually have had a coronary angioplasty or bypass procedure performed or have experienced an acute myocardial infarction shortly before death. In patients with acute infarction preceded by unstable angina pectoris, intracoronary lesions may not be representative of those occurring in patients with unstable angina not complicated by acute infarction.

Information regarding the acute coronary lesions in patients who died shortly after coronary artery bypass surgery has been provided in several studies. Guthrie et al. (10) described 12 patients and Roberts and Virmani (11) described 19 patients with unstable angina pectoris who died shortly after coronary bypass surgery. In both studies, the frequency of intraluminal thrombus was low (8% and 12%, respectively) when patients with acute myocardial infarction were excluded. In a separate report, Virmani and Roberts (12) described the frequency of extravasated erythrocytes and fibrin in the plaque of 17 of the 22 patients with unstable angina. Plaque hemorrhage (erythrocytes with or without fibrin) was identified in 94% of their patients. It is likely that surgical manipulation of the epicardial coronary arteries was responsible for the plaque hemorrhage in many of these cases.

In a study of unstable angina with fatal outcome, Falk (13) provided information regarding the frequency of acute lesions in the epicardial coronary arteries of patients with sudden coronary death, unstable angina pectoris and acute myocardial infarction. He described necropsy findings in "... 25 patients, all of whom died of acute coronary thrombosis within 24 hours after the onset of acute symptoms." Of the 24 patients for whom clinical information was available, 15 clearly had, 2 had an equivocal history of and 7 did not have unstable angina pectoris. Of these 25 patients, 15 had coagulative necrosis (acute myocardial infarction), that, as determined histologically, was compatible with an age of <24 h. In these patients, he described lamellar thrombi (21 of 25 patients, including 14 of 15 with unstable angina pectoris) 81% of which were associated with plaque rupture and hemorrhage. Neither the frequency of plaque rupture nor the number of thrombotic episodes differed between the patients with and without unstable angina. Because all of these patients died suddenly (some with unstable angina and some with unstable angina complicated by acute infarction), the three ischemic syndromes cannot be analyzed individually.

Sudden Coronary Death With or Without Unstable Angina

Davies et al. (14) studied 90 patients who died suddenly outside the hospital within 6 h of the onset of pain "or other symptoms." The data were presented in a report entitled "Intramyocardial platelet aggregation in patients with unstable angina suffering sudden ischemic cardiac death." Of their 90 patients, 36 (40%) had chest or arm pain at some time in the 2 weeks preceding death. The history of chest pain was obtained by a coroner's police officer from the next of kin who had been living with the patient. Thus, the history was not obtained from the patient or a physician. None of the 90 patients had been admitted to a hospital with increasing chest pain. There was no information on any patient regarding the presence or absence of chest pain at rest. Thus, in none of the 90 patients was the type, location or severity of the pain known. Nevertheless, these patients were considered to have unstable angina pectoris. Necropsy in the 90 patients disclosed the following: 31 (30%) had nonocclusive intracoronary thrombus, 22 (24%) had sudden coronary death associated with "regional coagulative necrosis" (acute myocardial infarction) and 23 (25%) had nontransmural necrosis. Of the 36 patients with chest or arm pain at some time in the 2 weeks before death, 35 had plaque rupture identified in one of the major epicardial coronary arteries. Of the 54 without chest pain in the 2 weeks before death, 51 had plaque rupture. Thus, whether the patients in that study had unstable angina pectoris is unknown. Some probably did have unstable angina pectoris, but some clearly had acute myocardial infarction and the majority would

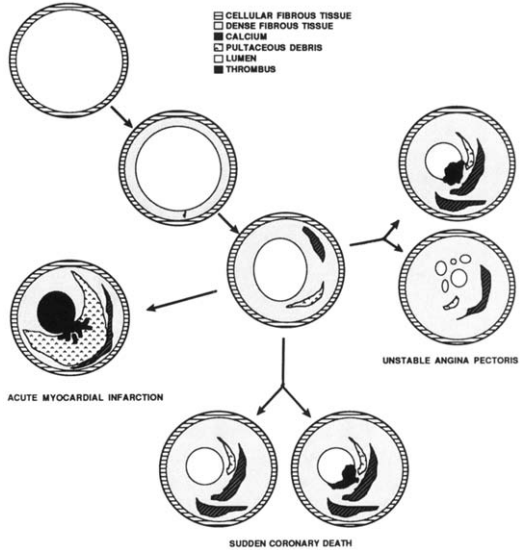

Figure 3. Diagram of coronary artery plaque morphology in patients with fatal coronary artery disease due to acute myocardial infarction, sudden coronary death without transmural left ventricular necrosis or unstable angina pectoris without transmural left ventricular necrosis. In all three groups, the mean percent of cellular fibrous tissue decreases with increasing degrees of luminal narrowing and the mean percent of dense fibrous tissue, calcific deposits and pultaceous debris rich in extracellular lipid increases. Severely narrowed segments in the group with acute myocardial infarction contain more pultaceous debris and are characterized by plaque rupture with associated hemorrhage and occlusive intraluminal thrombus. Severely narrowed segments in the group with unstable angina pectoris are characterized by the presence of multiluminal channels. Nonocclusive thrombus and plaque hemorrhage with or without plaque rupture can be seen in all three groups.

fulfill most investigators' definition of sudden coronary death. Diagnosing unstable angina pectoris in persons not admitted to the hospital whose history was obtained by a nonphysician is difficult to say the least.

Centrally placed multiluminal vascular channels. These vascular channels were present in 90% of our 67 patients. Most likely they represented organized thrombus (the consequence of a previous nonfatal thrombotic event) and were usually at a site where the lumen was severely narrowed by plaque. Multiluminal channels were observed in a significantly higher percent of the 5-mm coronary segments in our patients with unstable angina pectoris compared with those with either sudden coronary death or acute myocardial infarction. Data on the frequency of multiluminal channels in coronary plaque in patients with unstable angina, sudden death and acute infarction have been reported in only one previous study (12).

Plaque rupture and hemorrhage. It is possible that small sites of plaque rupture were not detected by examining the epicardial coronary arteries at 5-mm intervals in our study. It is likely, however, that associated plaque hemorrhage would have been detected. Although hemorrhage into plaque theoretically may be derived from sources other than intimal plaque rupture, the percent of cases that actually had plaque rupture (detected or not in this study) should be equal to or less than the percent of cases containing plaque hemorrhage. If this is the case, then the maximal percent of the unstable angina pectoris group that could have had plaque rupture is 64%; of the acute myocardial infarction group, 90% and of the sudden coronary death group, 38%. Even under these circumstances, plaque rupture would be much more common in acute infarction than in unstable angina or sudden death. Small nonocclusive thrombus or intraluminal platelet aggregates might also have been missed. Because all cases were handled in an identical fashion, it is likely that they would have been missed in all three groups with equal frequency.

Unstable angina and sudden coronary death versus acute myocardial infarction (Fig. 3). The lower frequency of plaque rupture and occlusive thrombus in the groups with unstable angina pectoris and sudden coronary death compared with that in the group with acute myocardial infarction may be a reflection of differences in plaque composition between these groups (16). Likewise, the similarity in the frequency of these acute coronary lesions in patients with unstable angina and sudden death may be a reflection of the similarity in plaque composition in these two groups (15). It has been shown (15,16) that in all three types of patients, the mean percent of dense fibrous tissue, calcific deposits and pultaceous debris increases with increasing degrees of lumi-

nal narrowing and the mean percent of cellular fibrous tissue decreases (Fig. 3). Severely narrowed segments in the acute myocardial infarction group (those narrowed >75% in cross-sectional area) contain significantly more pultaceous debris and significantly less calcium and cellular fibrous tissue than do similarly narrowed segments in the unstable angina pectoris and sudden coronary death groups. Because occlusive thrombus is almost exclusively seen in association with rupture of a lipid-rich plaque, the greater the amount of pultaceous debris, the greater is the frequency of plaque rupture and occlusive thrombus in these patients (16).

The characteristic lesion in patients with a fatal first acute myocardial infarction, then, is an occlusive thrombus overlying a ruptured plaque rich in pultaceous debris (Fig. 3); in patients with unstable angina pectoris, it is a severely narrowed segment frequently containing multiluminal channels with or without a small nonocclusive thrombus (Fig. 3); in patients with sudden coronary death without left ventricular necrosis, it is a segment of coronary artery with significant luminal narrowing by atherosclerotic plaque with or without platelet-rich nonocclusive thrombus (Fig. 3). Thus, the frequency of acute coronary lesions (intraluminal thrombus, plaque rupture and plaque hemorrhage) in patients with unstable angina (not complicated by acute myocardial infarction) and sudden death (not complicated by acute infarction) is similar and the frequency of these lesions is significantly lower than that observed in patients with acute infarction.

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